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PROBING SYNAPTIC AMYLOID-BETA AGGREGATION PROMOTED BY COPPER RELEASE

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Abstract:

Whether or not the metal ions released during synaptic transmission induce amyloid-beta oligomer formation in the vicinity of synapses is a central question pertinent to the molecular mechanism of Alzheimer's disease. Recently, through a combination of experimental kinetics studies and coupled reaction-diffusion simulations, we predicted that Cu(II) rather than Zn(II) plays an important role in the very early stages (i.e., dimer formation) of A β aggregation in the synapse. Single molecule photobleaching analysis is a powerful tool to determine the stoichiometry of amyloid-beta oligomers which enables us to examine the time course of small amyloid-beta oligomer formation in solution, immobilised to a solid-phase substrate or artificial lipid membrane, and in live neurons in the presence of Cu(II). Preliminary results indicate that small amyloid-beta oligomers can be locked in their oligomeric state without dissociation on a poly-lysine coated surface and that Cu(II) increases the diversity and abundance of amyloid-beta oligomers.

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